V 96

Cortical thickness and porosity assessment on ex-vivo tibiae using axial ultrasound transmission

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Osteoporosis is an underdiagnosed and undertreated metabolic bone disease. Currently, clinical fracture risk prediction is mainly based on a single parameter, i.e. bone mineral density (BMD). However, fracture resistance of bone is determined by a complex combination of micro-architectural, material and geometrical properties. Ultrasonic axial transmission (AT) measures the dispersion curves of guided waves (GWs) that propagate along the cortical layer of long bones, such as tibia and radius. Dispersion characteristics of GWs are determined by cortical thickness (C.Th) and mesoscopic stiffness, this latter material property depending strongly on cortical porosity. The goal of this study was to validate the cortical biomarkers at the tibia measured using AT. 20 tibiae from human cadavers were measured ex-vivo using a custom-made AT device (Azalée, Paris, France). Singular-value decomposition combined with 2D spatio-temporal Fourier transform was applied to extract the guided wave dispersion curves. C.Th and an index of cortical porosity (C.PI) were estimated after solving an inverse problem by fitting a 2D free transverse isotropic plate waveguide model to the experimental curves. Independent site-matched reference C.Th values were obtained from 39 µm voxel size high-resolution x-ray tomography (µCT). Reference C.PI was estimated from cross-sectional 100-MHz scanning acoustic microscopy (SAM) images. C.Th and C.PI were successfully obtained for 16 tibiae. The inverse problem could not be solved for 4 specimens due to poor ultrasonic response. Significant correlations ($p < 0.001$) were found between AT and the reference method (C.Th: $R2 = 0.84$, RMSE = 0.4 mm; C.PI: $R2 = 0.60$ RMSE = 3.8 %). For C.Th and C.PI biases of -0.12 mm and 3.41 % were observed, respectively. The cortical bone at the tibia was successfully characterized using AT ex-vivo. Further effort is now required to assess whether measurement of these bone strength related parameters enhance the prediction of atraumatic bone fractures.

V 98

The influence of gestational age on the maternal-foetal causal cardiac coupling

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Fetal development during pregnancy has been widely evaluated. Fetal heart rate variability (FHRV) is used as a reliable indicator of prenatal development. However, previous studies reported FHRV changes depending on the physiological and psychological states of the mother. However, the underlying mechanisms that generate FHRV patterns reflecting maternal–fetal cardiac couplings and their directionality are still poorly understood.
Therefore, the aim of this study was to quantify the direction of short-term maternal–fetal cardiac coupling in early, mid and late gestation fetuses by using the normalized short-time partial directed coherence (NSTPDC) analysis approach. We analyzed fetal electrocardiograms (fECGs) of 66 healthy fetuses; 22 from early gestation (16–25 weeks, GA1), 22 from mid gestation (26–30 weeks, GA2) and 22 from late gestation (32–41 weeks, GA3).
NSTPDC results demonstrated a causal influence of fetal on maternal heart rate in the early gestation, while it significantly decreased from early to mid-gestation age along with a significant increase of maternal to fetal coupling strength. The causal influence of maternal on fetal heart rate was the strongest in the mid gestation age and remained dominant in the late gestation. It seems to be that the maternal heart rate became a stronger driver for the fetal heart rate responses as time passes. This development begins in the second trimester and is especially pronounced in GA2.
In conclusion, we could demonstrate that the maternal–fetal cardiac coupling (strength and direction) between fetal and maternal heart rate changes with gestational age. In the mid gestation age the maternal to fetal coupling is dominated by the mother and retained strong afterwards.
This study provides detailed information about cardiac regulatory mechanisms in developing autonomic nervous system function in fetuses.

V 100

Screening for sleep apnea in routine Holter ECGs – a prospective evaluation

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Untreated sleep apnea is a risk factor for cardiovascular problems, and an economic burden for health care systems. This contribution presents an intermediate evaluation of the ongoing Carrera study („CARdiac REspitarory RAdar“). It targets accelerated identification of sleep apnea patients by prospective analysis of routine Holter-ECGs including typical complications like arrhythmias, co-medication, or comorbidity.
In 91 cardiologic in-patients (age: 64.3±12.1 years; BMI: 27.7±4.6kg/m2, 14 female) routinely scheduled for a Holter-ECG (Mortara H12+), we additionally registered a nocturnal polygraphy (PG, Heinen+Löwenstein Miniscreen 8). The apnea-/hypopnea index (AHI) and respiratory event index (REI) as obtained from the PG served as reference for an ECG-based severity index. This was quantified from modulations of ECG-amplitude and respiratory myogram interference based on correlation analysis. Agreement between PG- and ECG-based estimates was assessed using Bland-Altman diagrams with color-coded degree of ectopy. Moreover, accuracy of screening for REI≥15/h and AHI≥15/h was assessed.
The mean AHI was 16.0±14.7/h (median: 11.7/h), the average REI was 20.9±16.9/h (median: 16.3/h). We found a prevalence of 56% for REI ≥ 15/h, and a prevalence of 41% for AHI ≥ 15/h. 6 cases (7%) exhibited atrial fibrillation. The Bland-Altman diagrams indicated more consistent agreement of the ECG-based estimate with the REI. The agreement improved with better PG signal quality (i.e. lower sensor artifact time) and was better for lower REI/AHI values. In 75% of the cases the deviation was less than ±10 events/h. Reasons for deviations were identified. Arrhythmias did not derogate the estimate.
Screening for REI≥15/h yielded a specificity of 90% at 77% sensitivity. An alternative ternary screening strategy for AHI≥15/h identified 16% borderline cases with 82% sensitivity and 91% specificity for the remaining 84%.
We conclude that sleep-related breathing disorders have high prevalence in clinical cardiologic patient samples, and that screening of routine Holter-ECGs for sleep apnea is possible and reasonable.